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Search Results -

Terms	Documents
L1 same (purif? or isolat? or charact?)	12

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L2

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DATE: Tuesday, May 20, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L2	L1 same (purif? or isolat? or charact?)	12	L2
L1	(phospholipase A2)	1855	L1

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Search Results - Record(s) 1 through 12 of 12 returned.

1. Document ID: US 20030040088 A1

L2: Entry 1 of 12 File: PGPB Feb 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030040088
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030040088 A1

TITLE: Secreted protein HT5GJ57

PUBLICATION-DATE: February 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Ruben, Steven M.	Olney	MD	US	
Komatsoulis, George	Silver Spring	MD	US	
Duan, Roxanne D.	Bethesda	MD	US	
Rosen, Craig A.	Laytonsville	MD	US	
Moore, Paul A.	Germantown	MD	US	
Shi, Yanggu	Gaithersburg	MD	US	
LaFleur, David W.	Washington	DC	US	
Ebner, Reinhard	Gaithersburg	MD	US	
Olsen, Henrik S.	Gaithersburg	MD	US	
Brewer, Laurie A.	St. Paul	MN	US	
Florence, Kimberly A.	Rockville	MD	US	
Young, Paul E.	Gaithersburg	MD	US	
Mucenski, Michael	Cincinnati	OH	US	
Endress, Gregory A.	Florence	MA	US	
Soppet, Daniel R.	Centreville	VA	US	

US-CL-CURRENT: 435/183; 435/320.1, 435/325, 435/6, 435/69.1, 530/350, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

2. Document ID: US 20030017500 A1

L2: Entry 2 of 12 File: PGPB Jan 23, 2003

PGPUB-DOCUMENT-NUMBER: 20030017500
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030017500 A1

TITLE: Secreted protein HT5GJ57

PUBLICATION-DATE: January 23, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Ruben, Steven M.	Olney	MD	US	
Komatsoulis, George	Silver Spring	MD	US	
Duan, Roxanne D.	Bethesda	MD	US	
Rosen, Craig A.	Laytonsville	MD	US	
Moore, Paul A.	Germantown	MD	US	
Shi, Yanggu	Gaithersburg	MD	US	
LaFleur, David W.	Washington	DC	US	
Ebner, Reinhard	Gaithersburg	MD	US	
Olsen, Henrik	Gaithersburg	MD	US	
Brewer, Laurie A.	St. Paul	MN	US	
Florence, Kimberly A.	Rockville	MD	US	
Young, Paul	Gaithersburg	MD	US	
Mucenski, Michael	Cincinnati	OH	US	
Endress, Gregory A.	Florence	MA	US	
Soppet, Daniel R.	Centreville	VA	US	

US-CL-CURRENT: 435/7.1; 530/388.15, 530/388.26

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments

KWIC | Draw Desc | Image

☐ 3. Document ID: US 20020182652 A1

L2: Entry 3 of 12 File: PGPB Dec 5, 2002

PGPUB-DOCUMENT-NUMBER: 20020182652
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020182652 A1

TITLE: Proteomic analysis

PUBLICATION-DATE: December 5, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Cravatt, Benjamin F.	La Jolla	CA	US	
Sorensen, Erik	San Diego	CA	US	
Patricelli, Matthew P.	San Diego	CA	US	
Lovato, Martha	San Diego	CA	US	
Adam, Gregory	San Diego	CA	US	

US-CL-CURRENT: 435/7.9; 436/518

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments

KWIC | Draw Desc | Image

☐ 4. Document ID: US 20020086386 A1

L2: Entry 4 of 12 File: PGPB Jul 4, 2002

PGPUB-DOCUMENT-NUMBER: 20020086386
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020086386 A1

TITLE: B-catenin assays, and compositions therefrom

PUBLICATION-DATE: July 4, 2002

INVENTOR-INFORMATION: NAME	CITY	STATE	COUNTRY	RULE-47
Kamb, Carl Alexander	Salt Lake City	UT	US	
Yoo, Sanghee	Salt Lake City	UT	US	
Garcia-Guzman, Miguel	Salt Lake City	UT	US	
Pierce, Michael Leslie	Salt Lake City	UT	US	

US-CL-CURRENT: 435/183; 435/320.1, 435/325, 435/69.1, 530/350

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 5. Document ID: US 20020081688 A1

L2: Entry 5 of 12 File: PGPB Jun 27, 2002

PGPUB-DOCUMENT-NUMBER: 20020081688
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020081688 A1

TITLE: Retinoid pathway assays, and compositions therefrom

PUBLICATION-DATE: June 27, 2002

INVENTOR-INFORMATION: NAME	CITY	STATE	COUNTRY	RULE-47
Kamb, Carl Alexander	Salt Lake City	UT	US	
Richards, Burt Timothy	Midway	UT	US	
Karpilow, Jon	Boulder	CO	US	

US-CL-CURRENT: 435/189; 435/320.1, 435/325, 435/6, 435/69.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 6. Document ID: US 20020064799 A1

L2: Entry 6 of 12 File: PGPB May 30, 2002

PGPUB-DOCUMENT-NUMBER: 20020064799
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020064799 A1

TITLE: Proteomic analysis

PUBLICATION-DATE: May 30, 2002

INVENTOR-INFORMATION: NAME	CITY	STATE	COUNTRY	RULE-47
Cravatt, Benjamin F.	La Jolla	CA	US	
Sorensen, Erik	San Diego	CA	US	
Patricelli, Matthew P.	San Diego	CA	US	
Lovato, Martha	San Diego	CA	US	
Adam, Gregory	San Diego	CA	US	

US-CL-CURRENT: 435/7.1; 546/339, 548/570, 568/25

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 7. Document ID: US 20020045194 A1

L2: Entry 7 of 12 File: PGPB Apr 18, 2002

PGPUB-DOCUMENT-NUMBER: 20020045194
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020045194 A1

TITLE: Proteomic analysis

PUBLICATION-DATE: April 18, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Cravatt, Benjamin F.	La Jolla	CA	US	
Sorensen, Erik	San Diego	CA	US	
Patricelli, Matthew P.	San Diego	CA	US	
Lovato, Martha	San Diego	CA	US	
Adam, Gregory	San Diego	CA	US	

US-CL-CURRENT: 435/7.9

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 8. Document ID: US 20020040275 A1

L2: Entry 8 of 12 File: PGPB Apr 4, 2002

PGPUB-DOCUMENT-NUMBER: 20020040275
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020040275 A1

TITLE: Proteomic analysis

PUBLICATION-DATE: April 4, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Cravatt, Benjamin F.	La Jolla	CA	US	
Sorensen, Erik	San Diego	CA	US	
Patricelli, Matthew P.	San Diego	CA	US	
Lovato, Martha	San Diego	CA	US	
Adam, Gregory	San Diego	CA	US	

US-CL-CURRENT: 702/19; 435/7.1, 435/7.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 9. Document ID: US 6534631 B1

L2: Entry 9 of 12 File: USPT Mar 18, 2003

US-PAT-NO: 6534631
DOCUMENT-IDENTIFIER: US 6534631 B1

TITLE: Secreted protein HT5GJ57

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 10. Document ID: US 5229367 A

L2: Entry 10 of 12 File: USPT Jul 20, 1993

US-PAT-NO: 5229367
DOCUMENT-IDENTIFIER: US 5229367 A

TITLE: Antiinflammatory peptide derived from human lipocortin V

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 11. Document ID: JP 07011283 A

L2: Entry 11 of 12 File: JPAB Jan 13, 1995

PUB-NO: JP407011283A
DOCUMENT-IDENTIFIER: JP 07011283 A
TITLE: METHOD FOR PURIFYING OIL AND FAT

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 12. Document ID: JP 08168390 A

L2: Entry 12 of 12 File: DWPI Jul 2, 1996

DERWENT-ACC-NO: 1996-357246
DERWENT-WEEK: 199636

TITLE: Prepn of glyceride contg highly unsatd fatty acid in high concn - by contacting yolk phospholipid with phospholipase A2, adding glycerol to extracted fatty acid and reacting with lipase

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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Terms	Documents
L1 same (purif? or isolat? or charact?)	12

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11172	FILE BIOSIS
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              10   FILE SYNTHLINE
            9242   FILE TOXCENTER
             912   FILE USPATFULL
              26   FILE USPAT2
              18   FILE VETU
             643   FILE WPIDS
             643   FILE WPINDEX
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     FILE 'CAPLUS, EMBASE, BIOSIS, MEDLINE, TOXCENTER, SCISEARCH, BIOTECHNO,
     LIFESCI' ENTERED AT 13:09:07 ON 20 MAY 2003
L2          1954 S L1 AND (LIPOPROTEIN)
L3             0 S L2 AND PHOSPHOTIDYLCHOLINE
L4            10 S L2 AND (SN-2 ESTER)
L5             4 DUP REM L4 (6 DUPLICATES REMOVED)
L6           618 S L2 AND (ISOLAT? OR PURIF? OR CHARACT?)
L7             1 S L6 AND (SN-2 ESTER)
L8           267 S L6 AND PY<1993
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L8 ANSWER 264 OF 267 LIFESCI COPYRIGHT 2003 CSA
ACCESSION NUMBER: 90:65877 LIFESCI
TITLE: Enhanced binding of phospholipase-A sub(2)-modified low density **lipoprotein** by human adipocytes.
AUTHOR: Natarajan, M.K.; Fong, B.S.; Angel, A.
CORPORATE SOURCE: Rm. 7368, Med. Sci. Build., Univ. Toronto, Toronto, Ont. M5S 1A8, Canada
SOURCE: BIOCHEM. CELL BIOL., (**1990**) vol. 68, no. 11, pp. 1243-1249.
DOCUMENT TYPE: Journal
FILE SEGMENT: M
LANGUAGE: English
SUMMARY LANGUAGE: English; French
AB Recognition of low density **lipoprotein** (LDL) by human adipocytes is not dependent on the classical LDL (apoprotein B-E) receptor. To assess whether LDL phospholipids have a role in adipocyte-LDL interactions, binding studies were carried out with human LDL modified with cobra venom phospholipase A sub(2) (PLA sub(2)) and freshly **isolated** adipocytes and **purified** adipocyte plasma membranes prepared from surgical biopsies. LDL incubated with PLA sub(2) showed increased monoacylphospholipid content, decreased diacylphospholipid content, and increased anodic migration on agarose gel electrophoresis.

L8 ANSWER 263 OF 267 LIFESCI COPYRIGHT 2003 CSA
ACCESSION NUMBER: 91:58111 LIFESCI
TITLE: **Characterization** of several phospholipase activities and diacylglycerol/2-monoacylglycerol lipases in rat alveolar macrophages.
AUTHOR: Errasfa, M.
CORPORATE SOURCE: Dep. Prev. Med., Harvard Med. Sch., Massachusetts Gen. Hosp., Boston, MA 02114, USA
SOURCE: BIOCHIM. BIOPHYS. ACTA., (**1991**) vol. 1085, no. 2, pp. 201-208.
DOCUMENT TYPE: Journal
FILE SEGMENT: L
LANGUAGE: English
SUMMARY LANGUAGE: English
AB We measured phospholipase activities in both the microsomal and the cytosolic enriched fractions of rat alveolar macrophages by using exogenous arachidonic acid-labeled phospholipids. The largest release of arachidonic acid from PI occurred with the cytosolic fractions at pH 6 in the presence of calcium. That hydrolysis involved a PLA sub(2), and a PLC followed by the action of a diacylglycerol and 2-monoacylglycerol lipases. The cytosol also contains a calcium-independent PLA sub(2) acting on PE.

L8 ANSWER 258 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.

ACCESSION NUMBER: 1988:18171703 BIOTECHNO

TITLE: Lipolysis of LDL with phospholipase A.sub.2 alters the expression of selected apoB-100 epitopes and the interaction of LDL with cells

AUTHOR: Kleinman Y.; Krul E.S.; Burnes M.; Aronson W.; Pfleger B.; Schonfeld G.

CORPORATE SOURCE: Division of Atherosclerosis and Lipid Research, Department of Medicine, Washington University School of Medicine, St. Louis, MO 63110, United States.

SOURCE: Journal of Lipid Research, (**1988**), 29/6 (729-743)
CODEN: JLPRAW ISSN: 0022-2275

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB To assess the effects of perturbing the surface of low density **lipoprotein** (LDL) on the conformation of apoB-100, LDL (d 1.030-1.050 g/ml) **isolated** from normal subjects were treated with phospholipase A.sub.2 (PL-A.sub.2) for 0.5 to 15 min. The resulting P-LDL and concurrent LDL (C-LDL) incubated without PL-A.sub.2 were **isolated** by gel permeation chromatography. Approximately 50% of LDL-phosphatidylcholine was hydrolyzed in 2 min and .sim.85% in 5 min. Lysophosphatidylcholine compounds (LPC) and free fatty acids (FFA) accumulated during lipolysis but most of the LPC and all of FFA could be removed by adding FFA-free albumin to the lipolysis mixtures. Immunoreactivities of P-LDL and C-LDL were evaluated in competitive radioimmunoassays, using a library of anti-human LDL monoclonal antibodies directed against the major regions of apoB-100 (the T4, T3, and T2 thrombin fragments). One epitope defined by monoclonal antibody 465B6C3 and localized near the carboxyl end of the apoB-100 molecule became less immunoreactive (ED 50s increased); three other epitopes on the T2 fragment near the LDL receptor recognition site and four epitopes localized towards the middle (T3) and amino terminal (T4) regions did not change. Altered immunoreactivities were not related to LPC and FFA contents. Thus, the conformation of apoB-100 was selectively altered by phospholipolysis. The interactions of P-LDL with cultured fibroblasts were grossly altered: P-LDL were bound nonspecifically to fibroblasts of both normal and homozygous familial hypercholesterolemic subjects and P-LDL were not degraded. LPC and FFA retained in LDL did not explain these alterations, nor did changes of epitope expression near the LDL receptor recognition site. It is likely that the apoB-100 aberrant cell interaction is due to loss of surface phospholipids and 'uncovering' of core lipids that react nonspecifically with cell surface components.

L8 ANSWER 257 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
ACCESSION NUMBER: 1990:20043446 BIOTECHNO
TITLE: Apolipoprotein C-1 inhibits the hydrolysis by phospholipase A.sub.2 of phospholipids in liposomes and cell membranes
AUTHOR: Poensgen J.
CORPORATE SOURCE: Grunenthal GmbH, Aachen, Germany.
SOURCE: Biochimica et Biophysica Acta - Lipids and Lipid Metabolism, (**1990**), 1042/2 (188-192)
CODEN: BBLLA6 ISSN: 0005-2760
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English

AB A small polypeptide **isolated** from human serum inhibits the action of phospholipase A.sub.2 on dipalmitoylglycerol phosphocholine vesicles. Sequence analysis revealed the protein to be apolipoprotein C-1, a major component of very light-density **lipoprotein**. The inhibiting efficiency is increased by one order of magnitude after 10 min preincubation of the protein with the substrate, but not the enzyme. It also depends on the concentration of the phospholipid. IC.sub.5.sub.0 is about 0.5 .mu.M at 0.2 mM DPPC and 1 .mu.M at 1 mM DPPC. Apolipoprotein C-1 is also inhibitory in a more physiological system: in broken human leukemia cells (HL-60 cells) it inhibits the release by endogenous phospholipases of arachidonic acid from membrane phospholipids. The effective concentrations correspond to those found in the serum. It is concluded that apolipoprotein C-1 and similar phospholipid-binding proteins may act as phospholipase inhibitors by blocking the access to the substrate.

L8 ANSWER 254 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
ACCESSION NUMBER: 1991:21275537 BIOTECHNO
TITLE: **Characterization** of several phospholipase activities and diacylglycerol/2-monoacylglycerol lipases in rat alveolar macrophages
AUTHOR: Errasfa M.
CORPORATE SOURCE: Dept. of Preventive Medicine, Harvard Medical School, General Hospital,Boston, MA 02114, United States.
SOURCE: Biochimica et Biophysica Acta - Lipids and Lipid Metabolism, (**1991**), 1085/2 (201-208)
CODEN: BBLLA6 ISSN: 0005-2760
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We measured phospholipase activities in both the microsomal and the cytosolic enriched fractions of rat alveolar macrophages by using exogenous arachidonic acid-labeled phospholipids. The microsomal fractions contain a neutral calcium-independent phospholipase A.sub.2 (PLA.sub.2) which acts on substrates phosphatidylcholine (PC) and phosphatidylinositol (PI), a calcium-independent PLA.sub.2 acting on phosphatidylethanolamine (PE), and a neutral calcium-dependent PI-specific PLC. The cytosolic fractions contain calcium-dependent phospholipases: PLA.sub.2 that hydrolyses PC at alkaline pH, and a neutral PI-specific phospholipase C (PLC). The largest release of arachidonic acid from PI occurred with the cytosolic fractions at pH 6 in the presence of calcium. That hydrolysis involved a PLA.sub.2, and a PLC followed by the action of a diacyglycerol and 2-monoacylglycerol lipases. The cytosol also contains a calcium-independent PLA.sub.2 acting on PE. Our investigation shows that rat alveolar macrophages possess a number of phospholipases, as well as diacylglycerol and 2-monoacylglycerol lipases. The above enzymes could play an essential role in the remodeling of membrane phospholipids in resting cells, and the generation of physiologically active lipids in activated cells.

L8 ANSWER 250 OF 267 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 88:369870 SCISEARCH
THE GENUINE ARTICLE: P1047
TITLE: ENZYMATIC MODIFICATION OF LOW-DENSITY **LIPOPROTEIN** BY **PURIFIED** LIPOXYGENASE PLUS **PHOSPHOLIPASE-A2** MIMICS CELL-MEDIATED OXIDATIVE MODIFICATION
AUTHOR: SPARROW C P; PARTHASARATHY S; STEINBERG D (Reprint)
CORPORATE SOURCE: UNIV CALIF SAN DIEGO, SCH MED, DEPT MED, DIV ENDOCRINOL & METAB, M-013D, LA JOLLA, CA, 92093
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF LIPID RESEARCH, (**1988**) Vol. 29, No. 6, pp. 745-753.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 36

L8 ANSWER 251 OF 267 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 87:583957 SCISEARCH
THE GENUINE ARTICLE: K4290
TITLE: ENZYMATIC MODIFICATION OF LOW-DENSITY-**LIPOPROTEIN** BY **PURIFIED** LIPOXYGENASE PLUS **PHOSPHOLIPASE-A2**
AUTHOR: SPARROW C P (Reprint); PARTHASARATHY S; STEINBERG D
CORPORATE SOURCE: UNIV CALIF SAN DIEGO, LA JOLLA, CA, 92093
COUNTRY OF AUTHOR: USA
SOURCE: CIRCULATION, (**1987**) Vol. 76, No. 4, pp. 478.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: No References

L8 ANSWER 252 OF 267 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 87:502416 SCISEARCH
THE GENUINE ARTICLE: J8106
TITLE: ALPHA-LECITHIN - CHOLESTEROL ACYLTRANSFERASE DEFICIENCY - LACK OF BOTH **PHOSPHOLIPASE-A2** AND ACYLTRANSFERASE ACTIVITIES **CHARACTERISTIC** OF HIGH-DENSITY-**LIPOPROTEIN** LECITHIN - CHOLESTEROL ACYLTRANSFERASE IN FISH EYE DISEASE
AUTHOR: HOLMQUIST L (Reprint); CARLSON L A
CORPORATE SOURCE: KAROLINSKA INST, KING GUSTAV V RES INST, BOX 60004, S-10401 STOCKHOLM 60, SWEDEN (Reprint); KAROLINSKA INST, DEPT INTERNAL MED, S-10401 STOCKHOLM 60, SWEDEN; KAROLINSKA HOSP, S-10401 STOCKHOLM 60, SWEDEN
COUNTRY OF AUTHOR: SWEDEN
SOURCE: ACTA MEDICA SCANDINAVICA, (**1987**) Vol. 222, No. 1, pp. 23-26.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 12

L8 ANSWER 253 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
ACCESSION NUMBER: 1991:21310648 BIOTECHNO
TITLE: Factors affecting the ability of glycosylphosphatidylinositol-specific phospholipase D to degrade the membrane anchors of cell surface proteins
AUTHOR: Low M.G.; Huang K.-S.
CORPORATE SOURCE: Rover Physiology Laboratories, Department of Physiology, College of Physicians,New York, NY 10032,

United States.
SOURCE: Biochemical Journal, (**1991**), 279/2 (483-493)
CODEN: BIJOAK ISSN: 0264-6021
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Mammalian serum and plasma contain high levels of glycosylphosphatidylinositol-specific phospholipase D (GPI-PLD). Previous studies with crude serum or partially **purified** GPI-PLD have shown that this enzyme is capable of degrading the GPI anchor of several **purified** detergent-solubilized cell surface proteins yet is unable to act on GPI-anchored proteins located in intact cells. Treatment of intact ROS17/2.8, WISH or HeLa cells (or membrane fractions prepared from them) with GPI-PLD **purified** from bovine serum by immunoaffinity chromatography gave no detectable release of alkaline phosphatase into the medium. However, when membranes were treated with GPI-PLD in the presence of 0.1% Nonidet P-40 substantial GPI anchor degradation (as measured by Triton X-114 phase separation) was observed. The mechanism of this stimulatory effect of detergent was further investigated using .cents..sup.3H!myristate-labelled variant surface glycoprotein and human placental alkaline phosphatase reconstituted into phospholipid vesicles. As with the cell membranes the reconstituted substrates exhibited marked resistance to the action of **purified** GPI-PLD which could be overcome by the inclusion of Nonidet P-40. Similar results were obtained when crude bovine serum was used as the source of GPI-PLD. These data indicate that the resistance of cell membranes to the action of GPI-PLD is not entirely due to the action of serum or membrane-associated inhibitory factors. A more likely explanation is that, in common with many other eukaryotic phospholipases, the action of GPI-PLD is restricted by the physical state of the phospholipid bilayer in which the substrates are embedded. These data may account for the ability of endothelial and blood cells to retain GPI-anchored proteins on their surfaces in spite of the high levels of GPI-PLD present in plasma.

L8 ANSWER 254 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
ACCESSION NUMBER: 1991:21275537 BIOTECHNO
TITLE: **Characterization** of several phospholipase activities and diacylglycerol/2-monoacylglycerol lipases in rat alveolar macrophages
AUTHOR: Errasfa M.
CORPORATE SOURCE: Dept. of Preventive Medicine, Harvard Medical School, General Hospital,Boston, MA 02114, United States.
SOURCE: Biochimica et Biophysica Acta - Lipids and Lipid Metabolism, (**1991**), 1085/2 (201-208)
CODEN: BBLLA6 ISSN: 0005-2760
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English
AB We measured phospholipase activities in both the microsomal and the cytosolic enriched fractions of rat alveolar macrophages by using exogenous arachidonic acid-labeled phospholipids. The microsomal fractions contain a neutral calcium-independent phospholipase A.sub.2 (PLA.sub.2) which acts on substrates phosphatidylcholine (PC) and phosphatidylinositol (PI), a calcium-independent PLA.sub.2 acting on phosphatidylethanolamine (PE), and a neutral calcium-dependent PI-specific PLC. The cytosolic fractions contain calcium-dependent phospholipases: PLA.sub.2 that hydrolyses PC at alkaline pH, and a neutral PI-specific phospholipase C (PLC). The largest release of arachidonic acid from PI occurred with the cytosolic fractions at pH 6 in the presence of calcium. That hydrolysis involved a PLA.sub.2, and a PLC followed by the action of a diacyglycerol and 2-monoacylglycerol lipases. The cytosol also contains a calcium-independent PLA.sub.2 acting on PE.

Our investigation shows that rat alveolar macrophages possess a number of phospholipases, as well as diacylglycerol and 2-monoacylglycerol lipases. The above enzymes could play an essential role in the remodeling of membrane phospholipids in resting cells, and the generation of physiologically active lipids in activated cells.

L8 ANSWER 255 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
ACCESSION NUMBER: 1991:21212993 BIOTECHNO
TITLE: Conversion of pig pancreas phospholipase A.sub.2 by protein engineering into enzyme active against Escherichia coli treated with the bactericidal/permeability-increasing protein
AUTHOR: Weiss J.; Wright G.; Bekkers A.C.A.P.A.; Van den Bergh C.J.; Verheij H.M.
CORPORATE SOURCE: Dept. of Microbiology/Medicine, New York University, School of Medicine,New York, NY 10016, United States.
SOURCE: Journal of Biological Chemistry, (**1991**), 266/7 (4162-4167)
CODEN: JBCHA3 ISSN: 0021-9258
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Phospholipases A.sub.2 (PLA-2) are conserved enzymes that can vary widely in their activity toward certain biological targets. Activity of PLA-2 toward Escherichia coli treated with the bactericidal/permeability-increasing protein (BPI) of granulocytes has been detected only in ''Group II'' PLA-2 (lacking Cys.sup.1.sup.1-Cys.sup.7.sup.7) and correlates with overall basicity and the presence of a cluster of basic amino acids within a variable surface region near the NH.sub.2 terminus (including residues 6, 7, 10, 11, and 15). We now show that of five pancreatic PLA-2 (''Group I'' enzymes) tested from different species of mammals, the human enzyme that is most basic both globally (pI 8.7) and locally (Arg-6, Lys-7, and Lys-10) is active toward BPI-treated E. coli (.sim.1-2% activity of the most active Group II PLA-2) whereas the other four PLA-2 are essentially inactive (<0.1%). The cDNA of the pig pancreatic PLA-2 (pI 6.4; Arg-6, Ser-7, Lys-10) has been modified by site-specific mutagenesis and the wild-type and mutant PLA-2 have been expressed in and **purified** from either E. coli or Saccharomyces cerevisiae to determine more precisely the structural determinants of PLA-2 activity toward BPI-treated E. coli. The single substitution of lysine (or arginine) for Ser-7 transformed the pig pancreatic PLA-2 into an active enzyme toward BPI-treated E. coli possessing 25-50% the activity of the human PLA-2. Additional modifications to increase global basicity (increase in net charge up to +4) caused a further (up to 2-fold) increase in activity. All mutant PLA-2 still containing Ser-7 possessed little or no activity toward BPI-treated E. coli. Changes in activity toward BPI-treated E. coli were accompanied by parallel changes in enzyme binding to this target. In contrast, substitution of lysine (or arginine) for Ser-7 caused little or no alteration of enzyme activity toward either autoclaved E. coli or egg yolk **lipoproteins** indicating no major effects on the catalytic properties of the PLA-2. This study demonstrates directly the role of NH.sub.2-terminal basic residues in the action of PLA-2 on BPI-treated E. coli and suggests that these properties mainly facilitate PLA-2 binding to this biological target.

L8 ANSWER 256 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
ACCESSION NUMBER: 1991:21064584 BIOTECHNO
TITLE: Behaviour of phospholipase modified-HDL towards cultured hepatocytes. II. Increased cell cholesterol storage and bile acid synthesis
AUTHOR: Collet X.; Vieu C.; Chap H.; Perret B.-P.
CORPORATE SOURCE: INSERM Unite 326, Hopital Purpan,31059 Toulouse Cedex,

France.
SOURCE: Biochimica et Biophysica Acta - Lipids and Lipid Metabolism, (**1991**), 1081/2 (211-219)
CODEN: BBLLA6 ISSN: 0005-2760
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Human total HDL (hydrated density 1.070-1.210), HDL.sub.2 (1.070-1.125), HDL.sub.3 (1.125-1.210) or HDL separated by heparin affinity chromatography were treated with or without **purified** phospholipase A.sub.2 from Crotalus adamanteus. Control and treated HDL were reisolated and were then incubated with cultures hepatocytes. 1. Mass measurements evidenced a time-dependent cholesterol enrichment in hepatocytes cultured in the absence of **lipoproteins**. Addition of HDL.sub.2 still enhanced by 25% the cell cholesterol content and down-regulated endogenous sterol synthesis in similar proportions. Conversely, HDL.sub.3 slightly decreased the amount of free cholesterol in hepatocytes (-12%). 2. Incubations with phospholipase A.sub.2-treated HDL resulted in a 35%-50% increase of both the cellular cholesterol esterification and the cholesterylester accumulation, when compared to cells cultured in the presence of control-HDL. This effect was observed with HDL.sub.2, HDL.sub.3 and combining the data with all subfractions. 3. Cultured hepatocytes secreted cholic and .beta.-muricholic acids as major bile acids and HDL.sub.2 showed a tendency to stimulate their secretion. Phospholipase treatment of HDL again induced an increased production by hepatocytes of those two bile acids. Thus, whereas HDL.sub.2 and HDL.sub.3 display different behaviours with respect to cell cholesterol content, neosynthesis and bile acid secretion, their modifications by phospholipases always orientate the cell sterol metabolism in the same direction: increased cholesterylester accumulation and bile acid production.

L8 ANSWER 257 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
ACCESSION NUMBER: 1990:20043446 BIOTECHNO
TITLE: Apolipoprotein C-1 inhibits the hydrolysis by phospholipase A.sub.2 of phospholipids in liposomes and cell membranes
AUTHOR: Poensgen J.
CORPORATE SOURCE: Grunenthal GmbH, Aachen, Germany.
SOURCE: Biochimica et Biophysica Acta - Lipids and Lipid Metabolism, (**1990**), 1042/2 (188-192)
CODEN: BBLLA6 ISSN: 0005-2760
DOCUMENT TYPE: Journal; Article
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English

AB A small polypeptide **isolated** from human serum inhibits the action of phospholipase A.sub.2 on dipalmitoylglycerol phosphocholine vesicles. Sequence analysis revealed the protein to be apolipoprotein C-1, a major component of very light-density **lipoprotein**. The inhibiting efficiency is increased by one order of magnitude after 10 min preincubation of the protein with the substrate, but not the enzyme. It also depends on the concentration of the phospholipid. IC.sub.5.sub.0 is about 0.5 .mu.M at 0.2 mM DPPC and 1 .mu.M at 1 mM DPPC. Apolipoprotein C-1 is also inhibitory in a more physiological system: in broken human leukemia cells (HL-60 cells) it inhibits the release by endogenous phospholipases of arachidonic acid from membrane phospholipids. The effective concentrations correspond to those found in the serum. It is concluded that apolipoprotein C-1 and similar phospholipid-binding proteins may act as phospholipase inhibitors by blocking the access to the substrate.

L8 ANSWER 258 OF 267 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.

ACCESSION NUMBER: 1988:18171703 BIOTECHNO
TITLE: Lipolysis of LDL with phospholipase A.sub.2 alters the expression of selected apoB-100 epitopes and the interaction of LDL with cells
AUTHOR: Kleinman Y.; Krul E.S.; Burnes M.; Aronson W.; Pfleger B.; Schonfeld G.
CORPORATE SOURCE: Division of Atherosclerosis and Lipid Research, Department of Medicine, Washington University School of Medicine, St. Louis, MO 63110, United States.
SOURCE: Journal of Lipid Research, (**1988**), 29/6 (729-743)
CODEN: JLPRAW ISSN: 0022-2275
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB To assess the effects of perturbing the surface of low density **lipoprotein** (LDL) on the conformation of apoB-100, LDL (d 1.030-1.050 g/ml) **isolated** from normal subjects were treated with phospholipase A.sub.2 (PL-A.sub.2) for 0.5 to 15 min. The resulting P-LDL and concurrent LDL (C-LDL) incubated without PL-A.sub.2 were **isolated** by gel permeation chromatography. Approximately 50% of LDL-phosphatidylcholine was hydrolyzed in 2 min and .sim.85% in 5 min. Lysophosphatidylcholine compounds (LPC) and free fatty acids (FFA) accumulated during lipolysis but most of the LPC and all of FFA could be removed by adding FFA-free albumin to the lipolysis mixtures. Immunoreactivities of P-LDL and C-LDL were evaluated in competitive radioimmunoassays, using a library of anti-human LDL monoclonal antibodies directed against the major regions of apoB-100 (the T4, T3, and T2 thrombin fragments). One epitope defined by monoclonal antibody 465B6C3 and localized near the carboxyl end of the apoB-100 molecule became less immunoreactive (ED 50s increased); three other epitopes on the T2 fragment near the LDL receptor recognition site and four epitopes localized towards the middle (T3) and amino terminal (T4) regions did not change. Altered immunoreactivities were not related to LPC and FFA contents. Thus, the conformation of apoB-100 was selectively altered by phospholipolysis. The interactions of P-LDL with cultured fibroblasts were grossly altered: P-LDL were bound nonspecifically to fibroblasts of both normal and homozygous familial hypercholesterolemic subjects and P-LDL were not degraded. LPC and FFA retained in LDL did not explain these alterations, nor did changes of epitope expression near the LDL receptor recognition site. It is likely that the apoB-100 aberrant cell interaction is due to loss of surface phospholipids and 'uncovering' of core lipids that react nonspecifically with cell surface components.

L8 ANSWER 259 OF 267 BIOTECHNO
ACCESSION NUMBER: 1985:16194720 BIOTECHNO
TITLE: The degradation of platelet-activating factor in the plasma of a patient with familial high density **lipoprotein** deficiency (Tangier disease)
AUTHOR: Pritchard P.H.; Chonn A.; Yeung C.C.H.
CORPORATE SOURCE: Department of Pathology, Shaughnessy Hospital Lipid Research Group, The University of British Columbia, Vancouver, BC V6H 3N1, Canada.
SOURCE: Blood, (**1985**), 66/6 (1476-1478)
CODEN: BLOOAW
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English

AB Platelet Activating Factor (PAF) (1-O-alkyl-2-acetyl sn-glycerol 3-phosphocholine) has been **characterized** by its ability to aggregate platelets at low concentrations and its profound hypotensive effects. There is evidence that the rate of catabolism of this compound in the plasma regulates its concentration. In humans, we and others have

shown that a PAF acetylhydrolase is associated with low density **lipoprotein** (LDL). The LDL particle in the plasma of patients with Tangier disease is quite different from normal as its lipid core appears to be enriched with triacylglycerol. Thus, we have studied the potential of this abnormal **lipoprotein** to degrade PAF. The assay for PAF acetylhydrolase was based on the release of .sup.3H from PAF that was labelled in the acetate moiety of the sn-2 position. Tangier disease plasma had approximately 3.3-fold higher PAF acetylhydrolase activity (208 .+-. 9 nmol/min/mL) than controls (63 .+-. 18 nmol/min/mL). This increase was brought about by an increase in the Vmax (400 .+-. 40, Tangier disease; 54 .+-. 5, controls) and Km for PAF (120 .+-. 20 .mu.mol/L, Tangier disease; 28 .+-. 4 .mu.mol/L, controls). The activity appears to be a specific acetylhydrolase rather than a phospholipase A.sub.2 as preincubation of the substrate with 0 to 100 .mu.mol/L phosphatidylcholine did not affect the amount of .cents..sup.3H!acetate released. The role of PAF, and its degradation by LDL-bound PAF acetylhydrolase in the phenotypic expression of this patient with Tangier disease, is not known. However, this is the first patient so far described who has an increased ability to degrade PAF in the plasma.